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FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998

=> file uspatfull

=> s chronic renal failure

         17849 CHRONIC
          9598 RENAL
        176222 FAILURE
L1         380 CHRONIC RENAL FAILURE
                 (CHRONIC(W)RENAL(W)FAILURE)

=> s antiinflamm? or (anti inflammat?) or immunosuppress?

          5038 ANTIINFLAMM?
        156205 ANTI
         25237 INFLAMMAT?
         11801 ANTI INFLAMMAT?
                 (ANTI(W)INFLAMMAT?)
          4681 IMMUNOSUPPRESS?
L2       18112 ANTIINFLAMM? OR (ANTI INFLAMMAT?) OR
IMMUNOSUPPRESS?

=> s l1 and l2

L3          78 L1 AND L2

=> s (BONE(W)MORPHOGEN?) or BMP? or (OSTEOGENIC(W)(PROTEIN? OR
POLYPEPTIDE?))

         31383 BONE
           890 MORPHOGEN?
           438 BONE(W)MORPHOGEN?
           679 BMP?
           662 OSTEOGENIC
         78812 PROTEIN?
         18033 POLYPEPTIDE?
            94 OSTEOGENIC(W)(PROTEIN? OR POLYPEPTIDE?)
L4         845 (BONE(W)MORPHOGEN?) OR BMP? OR
(OSTEOGENIC(W)(PROTEIN? OR
                 POLYPEPTIDE?))

=> s TGFBETA## or (TGF(W)BETA##) or
((TRANSFORMING(W)GROWTH(W)FACTOR#)(1A)beta##)

             3 TGFBETA##
          1619 TGF
        171241 BETA##
          1044 TGF(W)BETA##
         27980 TRANSFORMING
        138446 GROWTH
        417281 FACTOR#
        171241 BETA##
           747 (TRANSFORMING(W)GROWTH(W)FACTOR#)(1A)BETA##
L5        1271 TGFBETA## OR (TGF(W)BETA##) OR
((TRANSFORMING(W)GROWTH(W)F
                 ACTOR#)(1A)BETA##)

=> s l2(p)l4

L6          26 L2(P)L4

=> s l2(p)l5

L7         109 L2(P)L5

=> d his


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     FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998
L1         380 S CHRONIC RENAL FAILURE
L2       18112 S ANTIINFLAMM? OR (ANTI INFLAMMAT?) OR
IMMUNOSUPPRESS?
L3            78 S L1 AND L2
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L5          1271 S TGFBETA## OR (TGF(W)BETA##) OR
((TRANSFORMING(W)GROWTH(
L6            26 S L2(P)L4
L7           109 S L2(P)L5

=> s l1 and 16

L8            0 L1 AND L6

=> s l1 and 17

L9            0 L1 AND L7

=> e glomerulo?

E1            2     GLOMERULNEPHRITIS/BI
E2           24     GLOMERULO/BI
E3            0 --> GLOMERULO?/BI
E4            1     GLOMERULOCAPSULAR/BI
E5            2     GLOMERULOID/BI
E6            1     GLOMERULOIDS/BI
E7            1     GLOMERULONE/BI
E8            1     GLOMERULONEHRITIS/BI
E9            1     GLOMERULONEPBRITIS/BI
E10           1     GLOMERULONEPHBRITIS/BI
E11           1     GLOMERULONEPHERITS/BI
E12           1     GLOMERULONEPHFITIS/BI

=> e

E13           4     GLOMERULONEPHITIS/BI
E14           1     GLOMERULONEPHRAL/BI
E15           1     GLOMERULONEPHRIS/BI
E16           1     GLOMERULONEPHRISIS/BI
E17           9     GLOMERULONEPHRITES/BI
E18           6     GLOMERULONEPHRITIC/BI
E19           7     GLOMERULONEPHRITIDES/BI
E20        1074     GLOMERULONEPHRITIS/BI
E21           1     GLOMERULONEPHRITISO/BI
E22           1     GLOMERULONEPHRITITS/BI
E23           3     GLOMERULONEPHRITUS/BI
E24           3     GLOMERULONEPHROPATHY/BI

=> e

E25           3     GLOMERULONEPHROSIS/BI
E26           3     GLOMERULONEPHTRITIS/BI
E27           1     GLOMERULONEPTHRITIS/BI
E28          19     GLOMERULOPATHIES/BI
E29          13     GLOMERULOPATHY/BI
E30           1     GLOMERULOPHROSIS/BI
E31           1     GLOMERULORSCLEROSIS/BI
E32          63     GLOMERULOSA/BI
E33          10     GLOMERULOSAL/BI
E34         114     GLOMERULOSCLEROSIS/BI
E35           1     GLOMERULOSCLEROSIS/BI
E36           2     GLOMERULOSCLEROTIC/BI

=> s e8-e29

             1 GLOMERULONEHRITIS/BI
             1 GLOMERULONEPBRITIS/BI
             1 GLOMERULONEPHBRITIS/BI
             1 GLOMERULONEPHERITS/BI
             1 GLOMERULONEPHFITIS/BI
             4 GLOMERULONEPHITIS/BI
             1 GLOMERULONEPHRAL/BI
             1 GLOMERULONEPHRIS/BI
             1 GLOMERULONEPHRISIS/BI
             9 GLOMERULONEPHRITES/BI
             6 GLOMERULONEPHRITIC/BI
             7 GLOMERULONEPHRITIDES/BI
          1074 GLOMERULONEPHRITIS/BI
             1 GLOMERULONEPHRITISO/BI
             1 GLOMERULONEPHRITITS/BI
             3 GLOMERULONEPHRITUS/BI
             3 GLOMERULONEPHROPATHY/BI
             3 GLOMERULONEPHROSIS/BI
             3 GLOMERULONEPHTRITIS/BI
             1 GLOMERULONEPTHRITIS/BI
            19 GLOMERULOPATHIES/BI
            13 GLOMERULOPATHY/BI
L10       1114 (GLOMERULONEHRITIS/BI OR GLOMERULONEPBRITIS/BI OR
GLOMERUL
                 ONEPHBRITIS/BI OR GLOMERULONEPHERITS/BI OR
GLOMERULONEPHFI
                 TIS/BI OR GLOMERULONEPHITIS/BI OR
GLOMERULONEPHRAL/BI OR
                 GLOMERULONEPHRIS/BI OR GLOMERULONEPHRISIS/BI OR
GLOMERULON
                 EPHRITES/BI OR GLOMERULONEPHRITIC/BI OR
GLOMERULONEPHRITID
                 ES/BI OR GLOMERULONEPHRITIS/BI OR
GLOMERULONEPHRITISO/BI
                 OR GLOMERULONEPHRITITS/BI OR GLOMERULONEPHRITUS/BI
OR
                 GLOMERULONEPHROPATHY/BI OR GLOMERULONEPHROSIS/BI
OR GLOMER
                 ULONEPHTRITIS/BI OR GLOMERULONEPTHRITIS/BI OR
GLOMERULOPAT
                 HIES/BI OR GLOMERULOPATHY/BI)

=> d his


     (FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

     FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998
L1         380 S CHRONIC RENAL FAILURE
L2       18112 S ANTIINFLAMM? OR (ANTI INFLAMMAT?) OR
IMMUNOSUPPRESS?
L3            78 S L1 AND L2
L4           845 S (BONE(W)MORPHOGEN?) OR BMP? OR
(OSTEOGENIC(W)(PROTEIN?
L5          1271 S TGFBETA## OR (TGF(W)BETA##) OR
((TRANSFORMING(W)GROWTH(
L6            26 S L2(P)L4
L7           109 S L2(P)L5
L8             0 S L1 AND L6
L9             0 S L1 AND L7
                 E GLOMERULO?
L10       1114 S E8-E29

=> s l10 and 16

L11           0 L10 AND L6

=> s l10 and 17

L12          15 L10 AND L7

=> select

ENTER ANSWER SET OR SMARTSELECT L# OR (L12):112

ENTER ANSWER NUMBER OR RANGE (1-):1-15

ENTER DISPLAY CODE (TI) OR ?:pn

E1 THROUGH E15 ASSIGNED

=> s e1-e15

             1 US5061786/PN
             1 US5268455/PN
             1 US5605930/PN
             1 US5635532/PN
             1 US5635533/PN
             1 US5654333/PN
             1 US5661179/PN
             1 US5688765/PN
             1 US5693607/PN
             1 US5693610/PN
             1 US5708025/PN
             1 US5710178/PN
             1 US5712307/PN
             1 US5747532/PN
             1 US5759836/PN
L13         15 (US5061786/PN OR US5268455/PN OR US5605930/PN OR
US5635532
                 /PN OR US5635533/PN OR US5654333/PN OR
US5661179/PN OR
                 US5688765/PN OR US5693607/PN OR US5693610/PN OR
US5708025/
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PN OR US5710178/PN OR US5712307/PN OR US5747532/PN OR US5759836/PN)

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L6 26 S L2(P)L4
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E GLOMERULO?
L10 1114 S E8-E29
L11 0 S L10 AND L6
L12 15 S L10 AND L7
SELECT L12 1-15 PN
L13 15 S E1-E15

=> s l13 and l7

L14 15 L13 AND L7

=> d kwic 1-15

L14 ANSWER 1 OF 15 USPATFULL
PI US 5759836 980602
<--
SUMM . . . Pharmacol. 216, 379-383, 1992). This is further substantiated by the observation that the upregulation of iNOS can be reduced by ***anti*** - ***inflammatory*** cytokines such as IL-4, IL-8, IL-10, ***TGF*** -. ***beta*** .-1, -2, and -3, and macrophage deactivating factor (Nussler and Billiar, 1993, supra). COX-2 is induced in a number of cell. . . (Lin et al., J. Biol. Chem. 264:17379-17383, 1989), IL-1 (Raz et al., Proc. Natl. Acad. Sci. USA 86:1657-1661, 1989) and ***TGF*** -. ***beta*** . (Bailey and Verma, Anal. Biochem. 196:11-18, 1991). PGE.sub.2 inhibits the production of cytokines (Ferreri et al., J. Biol. Chem. 267:9443-9449,. . .

L14 ANSWER 2 OF 15 USPATFULL
PI US 5747532 980505
<--
DETD . . . protein, bactericidal/permeability increasing protein, polymyxin B, and the like), inhibition of cytokine synthesis/release (e.g., employing phosphodiesterase inhibitors, IL-4, IL-10, IL-13, ***TGF*** -. ***beta*** ., corticosteroids, and the like), anti-cytokine therapy (e.g., employing antibodies to TNF, soluble TNF receptors, IL-1 receptor antagonists, antibodies to IL-1. . . and the like), inhibition of nitric oxide synthase enzymes (e.g., employing N-methyl-L-arginine, .epsilon.-N-iminoethyl-L-lysine, aminoguanidine, S-methyl isothiourea sulfate, and the like), ***immunosuppression*** (e.g., employing agents such as cyclosporin A, OKT3, FK506, and the like), diabetic therapy (e.g., employing agents such as free. . .

L14 ANSWER 3 OF 15 USPATFULL
PI US 5712307 980127
<--
DETD . . . profound changes in tumor behavior were accompanied by alterations in the expression of genes implicated in growth control, angiogenesis, and ***immunosuppression*** (e.g., TGF.alpha., HbF, and ***TGF*** -. ***beta*** .2).
DETD . . . athymic mice. Molecular analysis of brain and hormone-refractory prostate cancer cells revealed marked decline in the production and secretion of ***TGF*** . ***beta*** ., a protein implicated in growth control, angiogenesis, and ***immunosuppression*** . Treated prostatic cells exhibited decreased proteolytic activity mediated by urokinase-plasminogen activator, a molecular marker of disease progression in man.
DETD The malignant prostatic cell lines exhibit numerous abnormalities in gene expression, including increased production of autocrine tumor growth factor-.beta. (***TGF*** -. ***beta*** .) and elevated activity of urokinase plasminogen activator (uPA). Members of the ***TGF*** -. ***beta*** . family have been implicated in tumor growth control, angiogenesis, and ***immunosuppression*** . uPA, in contrast, is a serine protease involved in degradation of extracellular stroma and basal lamina structures, with the potential to facilitate tumor invasion and metastasis. It was of interest, therefore, to examine the effect of NaPA on ***TGF*** -. ***beta*** . and uPa expression in the prostatic tumor cells. Northern blot analysis of PC3 after 72 h treatment revealed a decrease in ***TGF*** -. ***beta*** .2 mRNA levels; the effect was specific for ***TGF*** -. ***beta*** .2 as there was no change in the expression of ***TGF*** -. ***beta*** .1. The decrease in ***TGF*** -. ***beta*** .2 was accompanied by approximately a twofold increase in the levels of HLA-A3 mRNA, as previously observed in treated human leukemic. . .

L14 ANSWER 4 OF 15 USPATFULL
PI US 5710178 980120
<--
DETD . . . profound changes in tumor behavior were accompanied by alterations in the expression of genes implicated in growth control, angiogenesis, and ***immunosuppression*** (e.g., TGF.alpha., HbF, and ***TGF*** -. ***beta*** .2).
DETD . . . athymic mice. Molecular analysis of brain and hormone-refractory prostate cancer cells revealed marked decline in the production and secretion of ***TGF*** . ***beta*** ., a protein implicated in growth control, angiogenesis, and ***immunosuppression*** . Treated prostatic cells exhibited decreased proteolytic activity mediated by urokinase-plasminogen activator, a molecular marker of disease progression in man.
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L14 ANSWER 5 OF 15 USPATFULL
PI US 5708025 980113
<--
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mRNA levels; the effect was specific for ***TGF*** -. ***beta*** .2 as there was no change in the expression of ***TGF*** -. ***beta*** .1. The decrease in ***TGF*** -.
beta .2 was accompanied by approximately a twofold increase
in the levels of HLA-A3 mRNA, as previously observed in treated
human leukemic. . .

L14 ANSWER 6 OF 15 USPATFULL
PI US 5693610 971202
<--
SUMM ***Transforming*** ***growth*** ***factor*** -. ***beta*** . (***TGF*** -. ***beta*** .) and
platelet-derived growth factor (PDGF) belong to such a growth
factor. ***TGF*** -. ***beta*** . was first found as a factor
to promote growth of a rat fibroblast. Thereafter, it has been
found that ***TGF*** -. ***beta*** . inhibits growth of a lot
of cells, strongly suppresses immunological activity, and
increases extracellular matrix. It is suggested that the
overproduction and/or abnormal metabolism of ***TGF*** -.
beta . are involved in various diseases and symptoms of
immunosuppression in a cancer patient or the like, fibroid
lung, hepatic fibrosis, glomerulonephritis, scleroderma, or the
like. Further, PDGF acts on. . .

L14 ANSWER 7 OF 15 USPATFULL
PI US 5693607 971202
<--
SUMM This invention relates to the fields of drug therapy and protein
synthesis. A soluble ***TGF*** -. ***beta*** . binding
protein fragment is used to treat conditions characterized by an
excess of ***TGF*** -. ***beta*** ., including
fibroproliferation and ***immunosuppression*** . The present
invention also relates to recombinant expression of the binding
protein fragment in prokaryotic and eukaryotic cells.
SUMM There have been several attempts to suppress the effects of
TGF -. ***beta*** . excess by administering antibody
which is specific for ***TGF*** -. ***beta*** .. In a pending
patent application Ser. No. 759,109, filed Sep. 6, 1991, now U.S.
Pat. No. 5,571,714 also assigned to Celtrix Pharmaceuticals, Inc.,
monoclonal antibodies to ***TGF*** -. ***beta*** . were shown
to have affinity constants ranging from 1.6.times.10.sup.7 L/mol
to 3.4.times.10.sup.8 L/mol in a competitive radioimmunoassay
test. These monoclonal antibodies were suggested for use in
treating tumor cells that produce ***TGF*** -. ***beta*** .
to counteract the ***immunosuppressive*** effects of
TGF -. ***beta*** .. Another proposed use was treating
metastatic cancers.
SUMM In another embodiment of the present invention, the method
provides for administration of ***TGF*** -. ***beta*** .
receptor fragment in the condition of ***TGF*** -. ***beta***
. excess characterized by ***immunosuppression*** associated
with an infectious disease. In a further embodiment, the
immunosuppression may be associated with trypanosomal
infection or viral infections such as human
immunosuppression virus, human T cell lymphotropic virus
(HTLV-1), lymphocytic choriomeningitis virus and hepatitis.
DRWD "A sufficient amount of ***TGF*** -. ***beta*** .-binding
receptor fragment" as used herein refers to the amount of
TGF -. ***beta*** . receptor fragment that neutralizes
the biologic activity of excess ***TGF*** -. ***beta*** .. It
may be determined by (1) suitable clinical variables of
improvement, (2) pathologic evaluation of the effects on fibrosis
and/or ***immunosuppression*** or prevention of fibrosis, or
(3) a direct inhibition of ***TGF*** -. ***beta*** ..
DRWD This invention provides for administering to an individual with a
medical condition associated with ***TGF*** -. ***beta*** .
excess a sufficient amount of ***TGF*** -. ***beta*** .-binding receptor fragment, such as s.beta.-RII, to reduce excess
TGF -. ***beta*** . activity in the individual. The
TGF -. ***beta*** .-binding receptor fragment is all or
only a portion of a receptor which is capable of binding
TGF -. ***beta*** .. s.beta.-RII is made by synthesizing
the extracellular domain of the ***TGF*** -. ***beta*** .
Type II receptor (.beta.-RII) and developing a fragment of this
.beta.-RII domain as a high affinity, soluble binding protein
(s.beta.-RII) for ***TGF*** . ***beta*** .. This invention
further provides for delivering s.beta.-RII to a site where
TGF -. ***beta*** . is in excess, such as in disease
states characterized by fibroproliferation and
immunosuppression such as is associated with infectious
disease.
DRWD . . . s.beta.-RII fragments of the present invention may be
used to treat viral infections in which there is an overproduction
of ***TGF*** -. ***beta*** . and ***immunosuppression*** .
Examples of viruses with which ***TGF*** -. ***beta*** .
excess is associated include, but are not limited to, hepatitis C,
lymphocytic choriomeningitis, human immunodeficiency virus (HIV),
and human T. . .
DRWD The s.beta.-RII of the present invention also may be used to
increase the efficacy of vaccines. Because ***TGF*** -.
beta . may cause ***immunosuppression*** , the
administration of s.beta.-RII can counteract
immunosuppression caused by ***TGF*** -. ***beta*** .
and increase the vaccine recipient's immune response to the
vaccine. s.beta.-RII should be particularly effective in
immunosuppressed patients. s.beta.-RII may be administered
before or concomitantly with the vaccine.
DRWD The medical history may reveal facts which support a diagnosis of
fibroproliferative disorder, collagen vascular disease,
immunosuppression , or of potential for problematic wound
healing, as in peritoneal adhesions following surgery, or
restenosis of blood vessels after coronary angioplasty. Conditions
which are identified as being associated with high levels of
TGF -. ***beta*** . and/or proliferation of fibrous
tissue are considered to cause ***TGF*** -. ***beta*** .
excess.
DETD Injection of SCW produces an acute inflammatory response which is
clinically detectable within hours and maximal in 3-5 days. When
anti- ***TGF*** -. ***beta*** . is injected directly into a
joint before ip administration of the SCW, inflammation at 24
hours is significantly below that observed in joints with the
irrelevant antibody. At the peak of the acute response,
inflammation of anti- ***TGF*** -. ***beta*** . joints remains
far below that of joints with the irrelevant antibody. Even if
joints are injected with anti- ***TGF*** -. ***beta*** . when
inflammation is well developed (day 13), anti- ***TGF*** -.
beta . still has a significant ***anti*** -
inflammatory effect, when compared to irrelevant antibody.
Because s.beta.-RII also binds ***TGF*** -. ***beta*** .,
s.beta.-RII has a similarly beneficial effect when given early or
late in the inflammatory process.

L14 ANSWER 8 OF 15 USPATFULL
PI US 5688765 971118
<--
SUMM . . . to be unique and lacrimal gland-specific. It also appears
that this hormone effect is not linked to a generalized, systemic
anti - ***inflammatory*** function. Building on these
new discoveries, the method of the invention involves a rejection
of the classical therapeutic approach to. . . effects of
systemic administration. Furthermore, since the androgen-induced
suppression of lacrimal gland inflammation could be mediated
through the induction of ***transforming*** ***growth***
factor -. ***beta*** . (***TGF*** -. ***beta*** .),
a potent ***immunosuppressive*** compound, local application
of ***TGF*** -. ***beta*** . should also have the same
effect.
DETD . . . be through the control of epithelial cell cytokine
production. In support of this hypothesis, as has been described
above, the ***anti*** - ***inflammatoryaction*** of
androgens in lacrimal tissue appears to be mediated not through
lymphocytes, but rather through epithelial cells. Moreover,
epithelial cells in other tissues are known to secrete numerous
cytokines, e.g., ***TGF*** -. ***beta*** . (137), and

also
serve in exocrine sites as active cellular participants in the
glandular inflammation in Sjogren's syndrome (138). In addition,
as will be described below, androgens increase the mRNA and
protein levels of the ***immunosuppressive*** cytokine,
TGF -. ***beta*** .1, in the lacrimal gland. This cytokine is thought to play a protective role in Sjogren's syndrome, and increased expression of ***TGF*** -. ***beta***
. mRNA has been correlated with reduced inflammation in salivary
glands of Sjogren's syndrome patients (129). In contrast, the
absence of ***TGF*** -. ***beta*** .1 leads to a pronounced
lymphocytic infiltration into both lacrimal and salivary glands
(139).
DETD . . . endocrine regulation of immune function in this tissue.
These studies, which were conducted with high stringency, RT-PCR
procedures, demonstrated that ***TGF*** -. ***beta*** .1,
TGF -. ***beta*** .2, ***TGF*** -. ***beta*** .3,
IL-6, TNF-.alpha. and IL-1.alpha. mRNA may be detected
consistently in lacrimal glands, as well as in isolated lacrimal
acinar epithelial. . . of male and female rats. As a corollary
to these studies, whether lacrimal glands of autoimmune mice
contain mRNAs for ***anti*** - ***inflammatory*** , as well
as pro-inflammatory, cytokines was also examined. This research,
which was performed with high stringency RT-PCR techniques, showed
that mRNA for IL-1.alpha., IL-1.beta., IL-2 receptor, IL-6,
TGF -. ***beta*** . and TNF-.alpha. may be detected
consistently in lacrimal glands of autoimmune female MRL/lpr mice.
The identity of these amplified products. . .
DETD . . . in lacrimal tissues of normal rats and autoimmune mice.
In addition, these results demonstrate that androgens stimulate
the accumulation of ***TGF*** -. ***beta*** .1 mRNA and
protein in the lacrimal gland. ***TGF*** -. ***beta*** .1, in
turn, is known to exert profound ***immunosuppressive***
activity, including the inhibition of T and B cell proliferation,
cytotoxic T cell generation, natural and lymphokine-activated
killing, T cell. . . production, and is believed to down
regulate inflammation in exocrine glands in Sjogren's syndrome
(129,139,140). Consequently, the androgen-induced increase in
TGF -. ***beta*** .1 could act to suppress lymphocytic
infiltration and to attenuate IL-1 and TNF-.alpha. production in
the lacrimal gland. These hormonal effects. . .

L14 ANSWER 9 OF 15 USPATFULL
PI US 5661179 970826
<--
DETD . . . profound changes in tumor behavior were accompanied by
alterations in the expression of genes implicated in growth
control, angiogenesis, and ***immunosuppression*** (e.g.,
TGF.alpha., HbF, and ***TGF*** -. ***beta*** .2).
DETD . . . athymic mice. Molecular analysis of brain and
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beta .,
a protein implicated in growth control, angiogenesis, and
immunosuppression . Treated prostatic cells exhibited
decreased proteolytic activity mediated by urokinase-plasminogen
activator, a molecular marker of disease progression in man.
DETD The malignant prostatic cell lines exhibit numerous abnormalities
in gene expression, including increased production of autocrine
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elevated activity of urokinase plasminogen activator (uPA).
Members of the ***TGF*** -. ***beta*** . family have been
implicated in tumor growth control, anglogenesis, and
immunosuppression . uPA, in contrast, is a serine protease
involved in degradation of extracellular stroma and basal lamina
structures, with the potential to facilitate tumor invasion and
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of NaPA on ***TGF*** -. ***beta*** . and uPa expression in
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h treatment revealed a decrease in ***TGF*** -.
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mRNA levels; the effect was specific for ***TGF*** -.
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TGF -. ***beta*** .1. The decrease in ***TGF***
-.
beta .2 was accompanied by approximately a twofold increase
in the levels of HLA-A3 mRNA, as previously observed in treated
human leukemic. . .

L14 ANSWER 10 OF 15 USPATFULL
PI US 5654333 970805
<--
DETD . . . profound changes in tumor behavior were accompanied by
alterations in the expression of genes implicated in growth
control, angiogenesis, and ***immunosuppression*** (e.g.,
TGF.alpha., HbF, and ***TGF*** -. ***beta*** .2).
DETD . . . in athymic mice. Molecular analysis of brain and
hormone-refractory prostate cancer cells revealed marked decline
in the production and secretion of ***TGF*** .
beta .,
a protein implicated in growth control, angiogenesis, and
immunosuppression . Treated prostatic cells exhibited
decreased proteolytic activity mediated by urokinase-plasminogen
activator, a molecular marker of disease progression in man.
DETD The malignant prostatic cell lines exhibit numerous abnormalities
in gene expression, including increased production of autocrine
tumor growth factor-.beta. (***TGF*** -. ***beta***
.) and
elevated activity of urokinase plasminogen activator (uPA).
Members of the ***TGF*** -. ***beta*** . family have been
implicated in tumor growth control, anglogenesis, and
immunosuppression . uPA, in contrast, is a serine protease
involved in degradation of extracellular stroma and basal lamina
structures, with the potential to facilitate tumor invasion and
metastasis. It was of interest, therefore, to examine the effect
of NaPA on ***TGF*** -. ***beta*** . and uPa expression in
the prostatic tumor cells. Northern blot analysis of PC3 after 72
h treatment revealed a decrease in ***TGF*** -.
beta .2
mRNA levels; the effect was specific for ***TGF*** -.
beta .2 as there was no change in the expression of
TGF -. ***beta*** .1. The decrease in ***TGF***
-.
beta .2 was accompanied by approximately a twofold

L14 ANSWER 11 OF 15 USPATFULL
PI US 5635533 970603
<--
DETD . . . profound changes in tumor behavior were accompanied by
alterations in the expression of genes implicated in growth
control, angiogenesis, and ***immunosuppression*** (e.g.,
TGF.alpha., HbF, and ***TGF*** -. ***beta*** .2).
DETD . . . athymic mice. Molecular analysis of brain and
hormone-refractory prostate cancer cells revealed marked decline
in the production and secretion of ***TGF*** .
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-.
beta .2 was accompanied by approximately a twofold

increase
in the levels of HLA-A3 mRNA, as previously observed in treated
human leukemic. . .

L14 ANSWER 12 OF 15 USPATFULL
PI US 5635532 970603
<--
DETD . . . profound changes in tumor behavior were accompanied by
alterations in the expression of genes implicated in growth
control, angiogenesis, and ***immunosuppression*** (e.g.,
TGF.alpha., HbF, and ***TGF*** -. ***beta*** .2).
DETD . . . athymic mice. Molecular analysis of brain and
hormone-refractory prostate cancer cells revealed marked decline
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Members of the ***TGF*** -. ***beta*** . family have been
implicated in tumor growth control, angiogenesis, and
immunosuppression . uPA, in contrast, is a serine protease
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of NaPA on ***TGF*** -. ***beta*** . and uPa expression in
the prostatic tumor cells. Northern blot analysis of PC3 after 72
h treatment revealed a decrease in ***TGF*** -. ***beta*** .2
mRNA levels; the effect was specific for ***TGF*** -.
beta .2 as there was no change in the expression of
TGF -. ***beta*** .1. The decrease in ***TGF*** -.
beta .2 was accompanied by approximately a twofold increase
in the levels of HLA-A3 mRNA, as previously observed in treated
human leukemic. . .

L14 ANSWER 13 OF 15 USPATFULL
PI US 5605930 970225
<--
DETD . . . profound changes in tumor behavior were accompanied by
alterations in the expression of genes implicated in growth
control, angiogenesis, and ***immunosuppression*** (e.g.,
TGF.alpha., HbF, and ***TGF*** -. ***beta*** .2).
DETD . . . athymic mice. Molecular analysis of brain and
hormone-refractory prostate cancer cells revealed marked decline
in the production and secretion of ***TGF*** . ***beta*** .,
a protein implicated in growth control, angiogenesis, and
immunosuppression . Treated prostatic cells exhibited
decreased proteolytic activity mediated by urokinase-plasminogen
activator, a molecular marker of disease progression in man.
DETD The malignant prostatic cell lines exhibit numerous abnormalities
in gene expression, including increased production of autocrine
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elevated activity of urokinase plasminogen activator (uPA).
Members of the ***TGF*** -. ***beta*** . family have been
implicated in tumor growth control, angiogenesis, and
immunosuppression . uPA, in contrast, is a serine protease
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metastasis. It was of interest, therefore, to examine the effect
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the prostatic tumor cells. Northern blot analysis of PC3 after 72
h treatment revealed a decrease in ***TGF*** -. ***beta*** .2
mRNA levels; the effect was specific for ***TGF*** -.
beta .2 as there was no change in the expression of
TGF -. ***beta*** .1. The decrease in ***TGF*** -.
beta .2 was accompanied by approximately a twofold increase
in the levels of HLA-A3 mRNA, as previously observed in treated
human leukemic. . .
DETD . . . (see FIG. 38); (b) over 90% decline in invasive capacity
(see FIG. 39); and (c) profound inhibition of expression of
TGF -. ***beta*** .2, coding for a 12.5-kD protein
implicated in glioma autocrine growth, angiogenesis, and
tumor-induced ***immunosuppression*** . Synergy between NaPA
and LOV could be due to the ability of each to block the MVA
pathway at distinct. . .
DETD The ***TGF*** . ***beta*** . autocrine growth regulatory
pathways are of particular interest in primary central nervous
system tumors. This pluripotential growth regulator is produced by
both primary malignant astrocytoma tissue (Clark WC, Bressler J:
TGF . ***beta*** .-like activity in tumors of the central
nervous system. J Neurosurg 68: 920-924, 1988; Samuels V, Barett
JM, Brochman Set al.:. . . Pathol 134: 895-902, 1989) and by
cell lines derived from such tumors (Jennings MT, Macina RJ,
Carver R et al.: ***TGF*** . ***beta*** .1 and ***TGF*** .
beta .2 are potential growth regulators for low grade and
malignant gliomas in vitro with evidence in support of an
autocrine hypothesis. Int. J. Cancer 49: 129-139, 1991). The
immunosuppressive effects of malignant astrocytoma cells
on cocultured lymphocytes in vitro has been convincingly linked to
TGF . ***beta*** . production and can be partially
neutralized by antibodies against ***TGF*** . ***beta*** ..
TGF . ***beta*** . has been shown to be a growth
regulator for gliomas in vitro (Jennings MT, Macina RJ, Carver R
et al.: ***TGF*** . ***beta*** .1 and ***TGF*** .
beta .2 are potential growth regulators for low grade and
malignant gliomas in vitro with evidence in support of an
autocrine hypothesis. Int. J. Cancer 49: 129-139, 1991). The role
of the ***TGF*** . ***beta*** . pathway in growth regulation
of medulloblastoma is less well established than for malignant
astrocytomas. The antiproliferative effect of ATRA on Daoy
medulloblastoma cells is associated with increased secretion of
TGF . ***beta*** .2 and with induction of ***TGF*** .
beta . receptor expression. Because the ***TGF*** .
beta . family of growth factors plays such an important
role in the biology of malignant astrocytomas, the initial focus
has been. . .

L14 ANSWER 14 OF 15 USPATFULL
PI US 5268455 931207
<--
AB A polypeptide is provided that excludes (a) a full-length mature
TGF -. ***beta*** . molecule or precursor ***TGF*** -.
beta . molecule or deletion variants of mature or precursor
TGF -. ***beta*** . molecules in which from about 1 to 10
amino acid residues have been deleted, (b) a polypeptide of the
sequence:. . . the sequence:
Arg-Asn-Leu-Glu-Glu-Asn-Cys-Cys-
Val-Arg-Pro-Leu-Tyr-Ile-Asp-Phe-Arg-Gln-Asp-Leu, the polypeptide
comprising an amino acid sequence that is based on conserved
sequences in the family of ***TGF*** -. ***beta*** .
molecules. Such polypeptides are particularly useful
therapeutically as ***immunosuppressive*** agents when coupled
to carrier proteins or crosslinked to form polymers.
SUMM ***TGF*** -. ***beta*** . has been shown to have numerous
regulatory actions on a wide variety of both normal and neoplastic
cells. Recent studies indicate an important role for ***TGF***
-. ***beta*** . in cells of the immune system (J. Kehrl et al.,
J. Exp. Med., 163:1037 [1986]; H-J. Ristow, Proc. Natl. Acad.. .
. (T. Matsui et al., Proc, Nail. Acad. Sci. U.S.A., 83:2438
[1986]; C. Shipley et al. Cancer Res., 46:2068 [1986]). Moreover,
TGF -. ***beta*** . has been described as a suppressor of
cytokine (e.g., IFN-.gamma., TNF-.alpha.) production, indicating
its use as an ***immunosuppressant*** for treating
inflammatory disorders (Espevik et al., J. Exp. Med., 166:571-576
[1987]; European Pat. Pub. No. 269,408 published Jun. 1,. .
issued Feb. 21, 1989), and as a promoter of cachexia (Beutler and
Cerami, New Eng. J. Med., 316:379 [1987]). Further, ***TGF***
-. ***beta*** . induces collagen secretion in human fibroblast
cultures (Roberts et al., Proc. Nat. Acad. Sci. USA 83:4167-4171

(1986); Chua et al.,. . .
SUMM . . . a novel class of immune modulators that are useful to
develop diagnostic assays for the presence in patient fluids of
immunosuppressive proteins such as ***TGF*** -.
beta ., or antibodies to such proteins.
SUMM In a still further embodiment, the invention comprises a method
for producing antibodies that neutralize ***immunosuppressive***
proteins comprising immunizing an animal with the polypeptide
identified above that has the sequence containing the X moiety and
isolating antibodies generated by the polypeptide that neutralize
at least one ***immunosuppressive*** protein, preferably
TGF -. ***beta*** ..
DETD As used herein, the term " ***immunosuppressive*** protein" as
used in the context that it is neutralized by antibodies generated
by the polypeptide herein generally refers to the protein to which
the polypeptide corresponds that exhibits
immunosuppressive activity. For example, a polypeptide
representing an internal, active sequence within the full-length
TGF -. ***beta*** . sequence would have ***TGF*** -.
beta . as its ***immunosuppressive*** protein to be
neutralized.
DETD "Biologically active" polypeptides herein are defined as those
having the ability to cross-react with antisera raised against
native ***TGF*** -. ***beta*** . (where native ***TGF***
-. ***beta*** . is that which is obtained from platelets or
other natural sources). Immunological crossreactivity is a measure
of a single active epitope and does not necessarily encompass an
active domain involved in ***immunosuppressive*** activity.
DETD . . . peptide neutralizes monoclonal and polyclonal antibodies
raised against a corresponding native protein known to have
biological/therapeutic activity, e.g., mature human ***TGF***
-. ***beta*** .. Other indications, specific for the ***TGF***
-. ***beta*** . polypeptide herein, include whether the peptide
stimulates release of PGE2 by IL-1 treated human fibroblasts,
interferes with the binding of full-length ***TGF*** -.
beta . to its receptors, or acts as an
immunosuppressive agent either in vitro or in vivo in an
animal model. The first step, then, involves determining that the
monomeric. . .
DETD Since the polypeptides herein are, in general, related to
immunosuppressive proteins, they are also useful as
imunogens to elicit antibodies capable of blocking the
immunosuppressive activity associated with or caused by
such ***immunosuppressive*** proteins, e.g.,
TGF -.
beta .. Examples of such activity include neoplastic or
viral disorders. For making immunogenic peptides capable of
eliciting antibodies to the ***immunosuppressive*** proteins,
the polypeptides are typically not ***immunosuppressive*** ,
either because they are in monomeric form or because they are
modified to be so. This modification can be performed by
substituting one or more of the amino acids within the polypeptide
polymer sequence to obtain non- ***immunosuppressive***
immunogenic forms of the polypeptides. The proper amino acids to
be modified can be tested simply by making the substitution. . .

L14 ANSWER 15 OF 15 USPATFULL
PI US 5061786 911029
<--
AB A polypeptide is provided that excludes (a) a full-length mature
TGF -. ***beta*** . molecule or precursor ***TGF*** -.
beta . molecule or deletion variants of mature or precursor
TGF -. ***beta*** . molecules in which from about 1 to 10
amino acid residues have been deleted, (b) a polypeptide of the
sequence:. . . sequence: Arg Asn-Leu-Glu-Glu-Asn-Cys-Cys-Val-
Arg-Pro-Leu-Tyr-Ile-Asp-Phe-Arg-Gln-Asp-Leu, said polypeptide
comprising an amino acid sequence that is based on conserved
sequences in the family of ***TGF*** -. ***beta*** .
molecules. Such polypeptides are particularly useful
therapeutically as ***immunosuppressive*** agents when coupled
to carrier proteins or crosslinked to form polymers.
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regulatory actions on a wide variety of both normal and neoplastic
cells. Recent studies indicate an important role for ***TGF***
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J. Exp. Med. 163:1037 [1986]; H-J Ristow, Proc. Natl. Acad.. . .
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its use as an ***immunosuppressant*** for treating
inflammatory disorders (Espevik et al., J. Exp. Med. 166: 571-576
[1987]; European Pat. Pub. No. 269,408 published June. . Feb.
21, 1989), and as a promoter of cachexia (Beutler and Cerami, New
Eng. J. Med., 316: 379 [1987]). Further, ***TGF*** -.
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cultures (Roberts et al., Proc. Nat. Acad. Sci. USA 83: 4167-4171
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SUMM . . . a novel class of immune modulators that are useful to
develop diagnostic assays for the presence in patient fluids of
immunosuppressive proteins such as ***TGF*** -.
beta ., or antibodies to such proteins.
SUMM In a still further embodiment, the invention comprises a method
for producing antibodies that neutralize ***immunosuppressive***
proteins comprising immunizing an animal with the polypeptide
identified above that has the sequence containing the X moiety and
isolating antibodies generated by the polypeptide that neutralize
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immunosuppressive activity. For example, a polypeptide
representing an internal, active sequence within the full-length
TGF -. ***beta*** . sequence would have ***TGF*** -.
beta . as its ***immunosuppressive*** protein to be
neutralized.
DETD "Biologically active" polypeptides herein are defined as those
having the ability to cross-react with antisera raised against
native ***TGF*** -. ***beta*** . (where native ***TGF***
-. ***beta*** . is that which is obtained from platelets or
other natural sources). Immunological cross-reactivity is a
measure of a single active epitope and does not necessarily
encompass an active domain involved in ***immunosuppressive***
activity.
DETD . . . peptide neutralizes monoclonal and polyclonal antibodies
raised against a corresponding native protein known to have
biological/therapeutic activity, e.g., mature human ***TGF***
-. ***beta*** .. Other indications, specific for the ***TGF***
-. ***beta*** . polypeptide herein, include whether the peptide
stimulates release of PGE2 by IL-1 treated human fibroblasts,
interferes with the binding of full-length ***TGF*** -.
beta . to its receptors, or acts as an
immunosuppressive agent either in vitro or in vivo in an
animal model. The first step, then, involves determining that the
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DETD Since the polypeptides herein are, in general, related to
immunosuppressive proteins, they are also useful as
immunogens to elicit antibodies capable of blocking the
immunosuppressive activity associated with or caused by
such ***immunosuppressive*** proteins, e.g.,
TGF -.
beta .. Examples of such activity include neoplastic or
viral disorders. For making immunogenic peptides capable of
eliciting antibodies to the ***immunosuppressive*** proteins,
the polypeptides are typically not ***immunosuppressive*** ,
either because they are in monomeric form or because they are
modified to be so. This modification can be performed by
substituting one or more of the amino acids within the polypeptide
polymer sequence to obtain non- ***immunosuppressive***
immunogenic forms of the polypeptides. The proper amino

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acids to
      be modified can be tested simply by making the
substitution.  .  .

=> d his


     (FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

     FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998
L1          380 S CHRONIC RENAL FAILURE
L2        18112 S ANTIINFLAMM? OR (ANTI INFLAMMAT?) OR
IMMUNOSUPPRESS?
L3           78 S L1 AND L2
L4          845 S (BONE(W)MORPHOGEN?) OR BMP? OR
(OSTEOGENIC(W)(PROTEIN?
L5         1271 S TGFBETA## OR (TGF(W)BETA##) OR
((TRANSFORMING(W)GROWTH(
L6           26 S L2(P)L4
L7          109 S L2(P)L5
L8            0 S L1 AND L6
L9            0 S L1 AND L7
              E GLOMERULO?
L10        1114 S E8-E29
L11           0 S L10 AND L6
L12          15 S L10 AND L7
               SELECT L12 1-15 PN
L13          15 S E1-E15
L14          15 S L13 AND L7

=> s l13 and l10

L15          15 L13 AND L10

=> d kwic 1-15


L15  ANSWER 1 OF 15  USPATFULL
PI     US 5759836  980602
 <--
SUMM   .  .  .  NO, and also decreasing the levels of PGEs. In
the
       MRL-lpr/lpr mutant mouse, NOS inhibitors prevent anti-DNA
immune
       complex and   ***glomerulonephritis***  , and reduce the
intensity
       of inflammatory arthritis (Weinberg et al., J. Exp. Med.
       179:651-660, 1994).

L15  ANSWER 2 OF 15  USPATFULL
PI     US 5747532  980505
 <--
DETD   .  .  .  disease), diabetes, arthritis, asthma,
Alzheimer's
       disease, Parkinson's disease, multiple sclerosis,
cirrhosis,
       allograft rejection, encephalomyelitis, meningitis,
pancreatitis,
       peritonitis, vasculitis, lymphocytic choriomeningitis,
       ***glomerulonephritis***  , uveitis, ileitis, liver
inflammation,
       renal inflammation, hemorrhagic shock, anaphylactic shock,
burn,
       infection (including bacterial, viral, fungal and
parasitic
       infections), hemodialysis,.  .  .

L15  ANSWER 3 OF 15  USPATFULL
PI     US 5712307  980127
 <--
DETD   .  .  .  including (1) non-malignant disorders associated
with
       abnormal differentiation programs, autoimmunity and
inflammatory
       processes, e.g., rheumatoid arthritis, Castleman's
disease,
       mesangial proliferation,   ***glomerulonephritis***  ,
uveitis,
       sepsis, autoimmune diseases such as lupus, inflammatory
bowel,
       type I diabetes, vasculitis, and several skin disorders of
cell
       differentiation.  .  .

L15  ANSWER 4 OF 15  USPATFULL
PI     US 5710178  980120
 <--
DETD   .  .  .  including (1) non-malignant disorders associated
with
       abnormal differentiation programs, autoimmunity and
inflammatory
       processes, e.g., rheumatoid arthritis, Castleman's
disease,
       mesangial proliferation,   ***glomerulonephritis***  ,
uveitis,
       sepsis, autoimmune diseases such as lupus, inflammatory
bowel,
       type I diabetes, vasculitis, and several skin disorders of
cell
       differentiation.  .  .

L15  ANSWER 5 OF 15  USPATFULL
PI     US 5708025  980113
 <--
DETD   .  .  .  including (1) non-malignant disorders associated
with
       abnormal differentiation programs, autoimmunity and
inflammatory
       processes, e.g., rheumatoid arthritis, Castleman's
disease,
       mesangial proliferation,   ***glomerulonephritis***  ,
uveitis,
       sepsis, autoimmune diseases such as lupus, inflammatory
bowel,
       type I diabetes, vasculitis, and several skin disorders of
cell
       differentiation.  .  .

L15  ANSWER 6 OF 15  USPATFULL
PI     US 5693610  971202
 <--
SUMM   .  .  .  are involved in various diseases and symptoms of
       immunosuppression in a cancer patient or the like, fibroid
lung,
       hepatic fibrosis,   ***glomerulonephritis***  ,
scleroderma, or
       the like. Further, PDGF acts on smooth muscle cells,
fibroblasts,
       nerve gliacytes or the like to promote their.  .  .
CLM    What is claimed is:
 .  .  .  and which contains about 18 to 38% by weight of
protein, said
       disease or condition being fibroid lung, hepatic fibrosis,
       ***glomerulonephritis***  , or scleroderma.


L15  ANSWER 7 OF 15  USPATFULL
PI     US 5693607  971202
 <--
SUMM   .  .  .  degrade these proteins. Thus, TGF-.beta. can
cause
       fibrous tissue to accumulate. For example, in diabetic
nephropathy
       and human mesangial proliferative
***glomerulonephritis***  ,
       both fibroproliferative diseases, a prominent and
important
       pathological feature is the accumulation of mesangial
matrix
       (Mauer et al. (1984) J..  .  .
SUMM   Border et al. (1990) Nature 346:371-74, found that
antiserum
       against TGF-.beta. suppressed experimentally induced
       ***glomerulonephritis***  , which was characterized by
mesangial
       proliferation. Border et al. reported that the antibodies
to
       TGF-.beta. which were raised in rabbits.  .  .
SUMM   Another way of suppressing TGF-.beta. in experimental
       ***glomerulonephritis***   in rats, which is associated
with
       TGF-.beta.1 excess, was a low-protein diet. Both the
excreted
       nitrogen and the expressed TGF-.beta.1.  .  .
SUMM   .  .  .  hepatic, intraocular and pulmonary fibrosis. In a
further
       embodiment, the TGF-.beta. receptor fragment is
administered to
       patients with diabetic nephropathy,
***glomerulonephritis***  ,
       proliferative vitreoretinopathy, rheumatoid arthritis,
liver
       cirrhosis, and biliary fibrosis.
DRWD   .  .  .  administration of s.beta.-RII fragments of the
present
       invention may be used in fibroproliferative disorders. As
       mentioned above, animal models of
***glomerulonephritis***
       have shown good results with anti-TGF-.beta. antibodies
blocking
       excess TGF-.beta.. These antibodies will be difficult to
deliver
       because they have.  .  .  Thus, it would be preferable to
       administer a lower molecular weight, native protein or
close
       analog, such as s.beta.-RII, in   ***glomerulonephritis***
.
       Kidney diseases associated with TGF-.beta. excess include,
but are
       not limited to, mesangial proliferative
***glomerulonephritis***
       , crescentic   ***glomerulonephritis***  , diabetic
nephropathy,
       renal interstitial fibrosis, renal fibrosis in transplant
patients
       receiving cyclosporin, and HIV-associated nephropathy.
These
       conditions are associated with.  .  .
DRWD   Systemic administration is the preferred mode of
administration in
       ***glomerulonephritis***  , liver cirrhosis,
immunosuppressive
       conditions (such as viral infections, AIDS and
trypanosomal
       infections), and in widespread skin diseases (such as
progressive.
       .  .
DETD   The effect of s.beta.-RII is compared with that of
anti-TGF
       antibody in a   ***glomerulonephritis***   model.
Experimental
       ***glomerulonephritis***   can be induced in rats with a
single
       injection of antithymocyte serum because the glomerular
mesangial
       cells express a thy-1.1 epitope on their surfaces. The
       experimental lesion is acute mesangial proliferative
       ***glomerulonephritis***   and is characterized by
expansion of
       the mesangial matrix and hypercellularity. The injured
cells also
       express more TGF-.beta.1 mRNA and.  .  .
DETD   First,   ***glomerulonephritis***   is induced in rats by
an
       intravenous injection of antithymocyte serum. Next, for
six days,
       three groups of rats are.  .  .
DETD   .  .  .  the kidneys, which are stained with periodic
acid-Schiff
       solution to emphasize the pathological changes. The
negative
       control kidneys have full-blown   ***glomerulonephritis***
with
       reddish-pink amorphous fibrous material filling most of
the
       glomerulus. The positive control kidneys have a staining
pattern
       which is.  .  .
CLM    What is claimed is:
       5. The method of claim 3 wherein said fibroproliferative
disorder
       is selected from the group consisting of diabetic
```

nephropathy,
glomerulonephritis , proliferative vitreoretinopathy, liver
cirrhosis, biliary fibrosis, and myelofibrosis.

L15 ANSWER 8 OF 15 USPATFULL
PI US 5688765 971118
<--
DETD . . . S., Goodman, J. R., and Siiteri, P. K., Effect of castration and sex-hormone treatment on survival, anti-nucleic
acid antibodies, and ***glomerulonephritis*** in NZB/NZW F1
mice. J. Exp. Med. 147:1568-1583 (1978).

L15 ANSWER 9 OF 15 USPATFULL
PI US 5661179 970826
<--
DETD . . . including (1) non-malignant disorders associated with
abnormal differentiation programs, autoimmunity and inflammatory
processes, e.g., rheumatoid arthritis, Castleman's disease,
mesangial proliferation, ***glomerulonephritis*** , uveitis,
sepsis, autoimmune diseases such as lupus, inflammatory bowel,
type I diabetes, vasculitis, and several skin disorders of cell
differentiation. . .

L15 ANSWER 10 OF 15 USPATFULL
PI US 5654333 970805
<--
DETD . . . including (1) non-malignant disorders associated with
abnormal differentiation programs, autoimmunity and inflammatory
processes, e.g., rheumatoid arthritis, Castleman's disease,
mesangial proliferation, ***glomerulonephritis*** , uveitis,
sepsis, autoimmune diseases such as lupus, inflammatory bowel,
type I diabetes, vasculitis, and several skin disorders of cell
differentiation. . .

L15 ANSWER 11 OF 15 USPATFULL
PI US 5635533 970603
<--
DETD . . . including (1) non-malignant disorders associated with
abnormal differentiation programs, autoimmunity and inflammatory
processes, e.g., rheumatoid arthritis, Castleman's disease,
mesangial proliferation, ***glomerulonephritis*** , uveitis,
sepsis, autoimmune diseases such as lupus, inflammatory bowel,
type I diabetes, vasculitis, and several skin disorders of cell
differentiation. . .

L15 ANSWER 12 OF 15 USPATFULL
PI US 5635532 970603
<--
DETD . . . including (1) non-malignant disorders associated with
abnormal differentiation programs, autoimmunity and inflammatory
processes, e.g., rheumatoid arthritis, Castleman's disease,
mesangial proliferation, ***glomerulonephritis*** , uveitis,
sepsis, autoimmune diseases such as lupus, inflammatory bowel,
type I diabetes, vasculitis, and several skin disorders of cell
differentiation. . .

L15 ANSWER 13 OF 15 USPATFULL
PI US 5605930 970225
<--
SUMM . . . of inhibiting may be used in a subject having any of the
following pathologies: rheumatoid arthritis, Castleman's disease,
mesangial proliferation, ***glomerulonephritis*** , uveitis,
sepsis, automimmunity inflammatory bowel, type I diabetes,
vasculitis and a cell differentiation associated skin disorder.
The inhibition, of course,. . .
DETD . . . of inhibiting may be used in a subject having any of the
following pathologies: rheumatoid arthritis, Castleman's disease,
mesangial proliferation, ***glomerulonephritis*** , uveitis,
sepsis, automimmunity inflammatory bowel, type I diabetes,
vasculitis and a cell differentiation associated skin disorder.
The inhibition, of course,. . .
DETD . . . including (1) non-malignant disorders associated with
abnormal differentiation programs, autoimmunity and inflammatory
processes, e.g., rheumatoid arthritis, Castleman's disease,
mesangial proliferation, ***glomerulonephritis*** , uveitis,
sepsis, autoimmune diseases such as lupus, inflammatory bowel,
type I diabetes, vasculitis, and several skin disorders of cell
differentiation. . .

L15 ANSWER 14 OF 15 USPATFULL
PI US 5268455 931207
<--
DETD . . . lupus erythematosus, rheumatoid arthritis, scleroderma,
dermatomyositis, polymyositis, unclassified connective diseases,
autoimmune hemolytic anemia, idiopathic thrombocytopenic purpura,
autoimmune thyroiditis, polyarteritis nodosum,
glomerulonephritis , uveitis, etc.

L15 ANSWER 15 OF 15 USPATFULL
PI US 5061786 911029
<--
DETD . . . lupus erythematosus, rheumatoid arthritis, scleroderma
dermatomyositis, polymyositis, unclassified connective diseases,
autoimmune hemolytic anemia, idiopathic thrombocytopenic purpura,
autoimmune thyroiditis, polyarteritis nodosum,
glomerulonephritis , uveitis, etc.

=> file medline

COST IN U.S. DOLLARS SINCE FILE
TOTAL ENTRY
SESSION
FULL ESTIMATED COST 17.85
18.30

FILE 'MEDLINE' ENTERED AT 17:24:48 ON 15 AUG 1998

FILE LAST UPDATED: 11 AUG 1998 (19980811/UP). FILE COVERS 1966 TO DATE.

THE MEDLINE FILE WAS RELOADED FEBRUARY 15, 1998, TO REFLECT THE ANNUAL
MESH (MEDICAL SUBJECT HEADING) CHANGES. ENTER HELP RLOAD FOR DETAILS.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

=> d his

(FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998
L1 380 S CHRONIC RENAL FAILURE
L2 18112 S ANTIINFLAMM? OR (ANTI INFLAMMAT?) OR IMMUNOSUPPRESS?
L3 78 S L1 AND L2
L4 845 S (BONE(W)MORPHOGEN?) OR BMP? OR (OSTEOGENIC(W)(PROTEIN?
L5 1271 S TGFBETA## OR (TGF(W)BETA##) OR ((TRANSFORMING(W)GROWTH(
L6 26 S L2(P)L4
L7 109 S L2(P)L5
L8 0 S L1 AND L6
L9 0 S L1 AND L7
E GLOMERULO?
L10 1114 S E8-E29
L11 0 S L10 AND L6
L12 15 S L10 AND L7
SELECT L12 1-15 PN
L13 15 S E1-E15
L14 15 S L13 AND L7
L15 15 S L13 AND L10

FILE 'MEDLINE' ENTERED AT 17:24:48 ON 15 AUG 1998

=> s l1 and l2

354059 CHRONIC
210701 RENAL
211454 FAILURE
10652 CHRONIC RENAL FAILURE
(CHRONIC(W)RENAL(W)FAILURE)
5575 ANTIINFLAMM?
263229 ANTI
154247 INFLAMMAT?
37381 ANTI INFLAMMAT?
(ANTI(W)INFLAMMAT?)
60980 IMMUNOSUPPRESS?
L16 232 L1 AND L2

=> s l16 and l4

277336 BONE
19348 MORPHOGEN?
1506 BONE(W)MORPHOGEN?
1262 BMP?
3474 OSTEOGENIC
1019390 PROTEIN?
66695 POLYPEPTIDE?
113 OSTEOGENIC(W)(PROTEIN? OR POLYPEPTIDE?)
L17 0 L16 AND L4

=> s l16 and l5

293 TGFBETA##
11091 TGF
327114 BETA##
8923 TGF(W)BETA##
24093 TRANSFORMING
467672 GROWTH
1400980 FACTOR#
327114 BETA##
11190 (TRANSFORMING(W)GROWTH(W)FACTOR#)(1A)BETA##
L18 1 L16 AND L5

=> d au ti so

L18 ANSWER 1 OF 1 MEDLINE
AU Shimizu T; Fukagawa M; Kuroda T; Hata S; Iwasaki Y; Nemoto M; Shirai

K; Yamauchi S; Margolin S B; Shimizu F; Kurokawa K
TI Pirfenidone prevents collagen accumulation in the remnant kidney in
rats with partial nephrectomy.
SO KIDNEY INTERNATIONAL. SUPPLEMENT, (1997 Dec) 63 S239-43.
Journal code: KVC. ISSN: 0098-6577.

=> d ab

L18 ANSWER 1 OF 1 MEDLINE
AB Pirfenidone (PFD) is a new compound that prevents and even reverses
the extracellular matrix accumulation in several organs as shown by
experimental and clinical studies. In the present study, we examined
the effect of PFD (500 mg/kg daily in the food) on the progression
of ***chronic*** ***renal*** ***failure*** (CRF) in the
5/6 nephrectomy rat model. Proteinuria progressively increased in
rats with renal ablation (C) at 12 weeks. Urinary protein excretion
in PFD-treated rats (P) was numerically lower than C, but the
difference did not reach statistical significance. In contrast, in
the chronic phase, PFD improved renal function and reduced collagen
accumulation detected by hydroxyproline content (OH-Pro) in the
cortex of the remnant kidney. Although creatinine clearance
decreased with time in C, the values in P were significantly better
at 10 and 12 weeks. The OH-Pro in C at 12 weeks was significantly
higher than that of no-ablation, sham-operated rats, whereas OH-Pro
in CRF was lower in (P). Expression of mRNA for type IV and I
collagen in the cortex also increased in C, but it was inhibited in
(P). To study the role that ***TGF*** - ***beta*** plays in
the regulatory process following CRF, we examined the expression of
TGF - ***beta*** mRNA in this model. Levels of cortical
TGF - ***beta*** mRNA in C were significantly elevated at
12 weeks. The increase was suppressed by PFD. These results
demonstrate that PFD attenuates the development of CRF by preventing
collagen accumulation in this model, and suggest that PFD can be
clinically useful for treating CRF.

=> d his

(FILE 'HOME' ENTERED AT 17:13:09 ON 15 AUG 1998)

FILE 'USPATFULL' ENTERED AT 17:14:49 ON 15 AUG 1998
L1 380 S CHRONIC RENAL FAILURE
L2 18112 S ANTIINFLAMM? OR (ANTI INFLAMMAT?) OR IMMUNOSUPPRESS?
L3 78 S L1 AND L2
L4 845 S (BONE(W)MORPHOGEN?) OR BMP? OR (OSTEOGENIC(W)(PROTEIN?
L5 1271 S TGFBETA## OR (TGF(W)BETA##) OR ((TRANSFORMING(W)GROWTH(
L6 26 S L2(P)L4
L7 109 S L2(P)L5
L8 0 S L1 AND L6
L9 0 S L1 AND L7
E GLOMERULO?
L10 1114 S E8-E29
L11 0 S L10 AND L6
L12 15 S L10 AND L7
SELECT L12 1-15 PN
L13 15 S E1-E15
L14 15 S L13 AND L7
L15 15 S L13 AND L10

FILE 'MEDLINE' ENTERED AT 17:24:48 ON 15 AUG 1998
L16 232 S L1 AND L2
L17 0 S L16 AND L4
L18 1 S L16 AND L5

=> file caplus

COST IN U.S. DOLLARS SINCE FILE
TOTAL ENTRY
SESSION
FULL ESTIMATED COST 2.50
20.80

FILE 'CAPLUS' ENTERED AT 17:29:42 ON 15 AUG 1998
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FILE COVERS 1967 - 15 Aug 1998 VOL 129 ISS 8
FILE LAST UPDATED: 15 Aug 1998 (980815/ED)

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=> s l16; s l19 and l4; s l19 and l5

91316 CHRONIC
81273 RENAL
88438 FAILURE
2950 CHRONIC RENAL FAILURE
(CHRONIC(W)RENAL(W)FAILURE)
28488 ANTIINFLAMM?
174425 ANTI
83266 INFLAMMAT?
14854 ANTI INFLAMMAT?
(ANTI(W)INFLAMMAT?)
25193 IMMUNOSUPPRESS?
L19 23 L1 AND L2

84342 BONE
12643 MORPHOGEN?
1542 BONE(W)MORPHOGEN?
1587 BMP?
1457 OSTEOGENIC
1119373 PROTEIN?
91655 POLYPEPTIDE?
146 OSTEOGENIC(W)(PROTEIN? OR POLYPEPTIDE?)
L20 0 L19 AND L4

846 TGFBETA##
11277 TGF
762216 BETA##
9132 TGF(W)BETA##
29410 TRANSFORMING
692464 GROWTH
758204 FACTOR#
762216 BETA##
11087 (TRANSFORMING(W)GROWTH(W)FACTOR#)(1A)BETA##
L21 0 L19 AND L5

=> d au ti so 1-23 l19

L19 ANSWER 1 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Tamimi, N. A.; Stevens, P. E.; O'Donnell, P. L.; Strange, P. G.;
Muchaneta-Kubara, E. C.; El Nahas, A. M.
TI Expression of cytoskeletal proteins differentiates between progressors and non-progressors in treated idiopathic membranous
nephropathy
SO Exp. Nephrol. (1998), 6(3), 217-225
CODEN: EXNEEG; ISSN: 1018-7782

L19 ANSWER 2 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Delzell, Elizabeth; Shapiro, Samuel
TI A review of epidemiologic studies of nonnarcotic analgesics and
chronic renal disease
SO Medicine (Baltimore) (1998), 77(2), 102-121
CODEN: MEDIAV; ISSN: 0025-7974

L19 ANSWER 3 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Fillastre, Jean-Paul
TI Ochratoxin-induced animal and human nephrotoxicity
SO Bull. Acad. Natl. Med. (Paris) (1997), 181(7), 1447-1463
CODEN: BANMAC; ISSN: 0001-4079

L19 ANSWER 4 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Takeuchi, H.; Hirano, T.; Oka, K.; Mizumoto, K.; Akashi, T.; Sakurai, E.; Degawa, T.; Uchiyama, M.; Kozaki, K.; Matsuno, N.;
Nagao, T.; Kozaki, M.
TI Lymphocyte sensitivity to cyclosporine and tacrolimus in ***chronic*** ***renal*** ***failure*** patients and
clinical significance in renal transplantation
SO Transplant. Proc. (1998), 30(1), 36-39
CODEN: TRPPA8; ISSN: 0041-1345

L19 ANSWER 5 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Smith, O. P.; White, B.; Vaughan, D.; Rafferty, M.; Claffey, L.;
Lyons, B.; Casey, W.
TI Use of protein-C concentrate, heparin, and hemodiafiltration in
Meningococcus-induced purpura fulminans
SO Lancet (1997), 350(9091), 1590-1593
CODEN: LANCAO; ISSN: 0140-6736

L19 ANSWER 6 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Morrone, Luigi F.; Paolo, Salvatore Di; Logoluso, Francesco; Schena,
Antonio; Stallone, Giovanni; Giorgino, Francesco; Schena, F. Paolo
TI Interference of angiotensin-converting enzyme inhibitors on erythropoiesis in kidney transplant recipients: role of growth
factors and cytokines
SO Transplantation (1997), 64(6), 913-918
CODEN: TRPLAU; ISSN: 0041-1337

L19 ANSWER 7 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Horigome, Atsushi; Hirano, Toshihiko; Oka, Kitaro; Takeuchi, Hironori; Sakurai, Etsuo; Kozaki, Koichi; Matsuno, Naoto; Nagao,
Takeshi; Kozaki, Masami
TI Glucocorticoids and cyclosporine induce apoptosis in mitogen-activated human peripheral mononuclear cells
SO Immunopharmacology (1997), 37(1), 87-94
CODEN: IMMUDP; ISSN: 0162-3109

L19 ANSWER 8 OF 23 CAPLUS COPYRIGHT 1998 ACS
IN Baker, Robert K.; Bao, Jianming; Kayser, Frank; Parsons, William H.;
Rupprecht, Kathleen M.
TI Triterpene derivatives with ***immunosuppressant*** activity,

their preparation, and compositions containing them
SO PCT Int. Appl., 121 pp.
CODEN: PIXXD2

L19 ANSWER 9 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Chan, Christopher; Maurer, Janet; Cardella, Carl; Cattran, Dan; Pei, York
TI A randomized controlled trial of verapamil on cyclosporine nephrotoxicity in heart and lung transplant recipients
SO Transplantation (1997), 63(10), 1435-1440
CODEN: TRPLAU; ISSN: 0041-1337

L19 ANSWER 10 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Murphy, Brendan G.
TI Lipoprotein (a) and the kidney
SO Nephrology (1997), 3(2), 139-142
CODEN: NEPHF2; ISSN: 1320-5358

L19 ANSWER 11 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Schulze-Lohoff, E.; Ogilvie, A.; Sterzel, R. B.
TI Extracellular nucleotides as signaling molecules for renal mesangial cells
SO J. Auton. Pharmacol. (1996), 16(6), 381-384
CODEN: JAPHDU; ISSN: 0144-1795

L19 ANSWER 12 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU De Lima, Jose J. G.; Maranhao, Raul C.; da Conceicao, Maria; Latrilha, M.; Diament, Jayme; Romao, Joao Egidio; Krieger, Eduardo M.; Pileggi, Fulvio
TI Early elevation of lipoprotein(a) levels in chronic renal insufficiency
SO Renal Failure (1997), 19(1), 145-154
CODEN: REFAE8; ISSN: 0886-022X

L19 ANSWER 13 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Faedda, Rossana; Pirisi, Mario; Satta, Andrea; Bosincu, Luisanna; Bartoli, Ettore
TI ***Immunosuppressive*** treatment of Berger's disease
SO Clin. Pharmacol. Ther. (St. Louis) (1996), 60(5), 561-567
CODEN: CLPTAT; ISSN: 0009-9236

L19 ANSWER 14 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Bonnin, Maria R.; Gonzalez, Maria T.; Grino, Jose M.; Cruzado, Jose M.; Martinez, Jose M.; Navarro, Miguel A.
TI Evolution of circulating C-terminal propeptide of type I procollagen in patients with ***chronic*** ***renal*** ***failure*** pre and post renal transplantation
SO Eur. J. Clin. Chem. Clin. Biochem. (1996), 34(11), 897-900
CODEN: EJCBEO; ISSN: 0939-4974

L19 ANSWER 15 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Elisaf, M.; Mikhailidis, D. P.; Siamopoulos, K. C.
TI Dyslipidemia in patients with renal diseases
SO J. Drug Dev. Clin. Pract. (1996), 7(4), 331-48
CODEN: JDCPFC; ISSN: 1357-9215

L19 ANSWER 16 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Stenvinkel, Peter; Berglund, Lars
TI Lipoprotein(a) in chronic renal disease
SO Miner. Electrolyte Metab. (1995), Volume Date 1996, 22(1-3), 16-21
CODEN: MELMDI; ISSN: 0378-0392

L19 ANSWER 17 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Kuroda, Minpei; Mimaki, Yoshihiro; Sashida, Yutaka; Hirano, Toshihiko; Oka, Kitaro; Dobashi, Akira
TI A novel 16,23-epoxy-5.beta.-cholestane glycoside with potent inhibitory activity on proliferation of human peripheral blood lymphocytes from Ornithogalum saundersiae bulbs
SO Chem. Pharm. Bull. (1995), 43(7), 1257-9
CODEN: CPBTAL; ISSN: 0009-2363

L19 ANSWER 18 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Agmon, Yoram; Brezis, Mayer
TI Effects of nonsteroidal ***anti*** - ***inflammatory*** drugs upon intrarenal blood flow: selective medullary hypoperfusion
SO Exp. Nephrol. (1993), 1(6), 357-63
CODEN: EXNEEG; ISSN: 1018-7782

L19 ANSWER 19 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Kinlen, L. J.
TI ***Immunosuppression*** and cancer
SO IARC Sci. Publ. (1992), 116(Mech. Carcinog. Risk Identif.), 237-53
CODEN: IARCCD; ISSN: 0300-5038

L19 ANSWER 20 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Ismail, F. A.; Khalafallah, N.; Khalil, Said A.
TI Disease and drug-induced changes in naproxen binding to plasma
SO Drug Dev. Ind. Pharm. (1989), 15(1), 147-64
CODEN: DDIPD8; ISSN: 0363-9045

L19 ANSWER 21 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Langhoff, E.; Ladefoged, J.; Odum, N.
TI Effect of interleukin-2 and methylprednisolone on in vitro transformation of uremic lymphocytes
SO Int. Arch. Allergy Appl. Immunol. (1986), 81(1), 5-11
CODEN: IAAAAM; ISSN: 0020-5915

L19 ANSWER 22 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Tvedegaard, Erling; Szpirt, Wladimir; Nielsen, Maja
TI Effect of ***chronic*** ***renal*** ***failure*** and methylprednisolone treatment on the uptake of labeled plasma cholesterol into the aorta of normocholesterolemic rabbits
SO Atherosclerosis (Shannon, Irel.) (1983), 47(2), 199-209
CODEN: ATHSBL; ISSN: 0021-9150

L19 ANSWER 23 OF 23 CAPLUS COPYRIGHT 1998 ACS
AU Schusziarra, V.; Ziekursch, V.; Schlamp, R.; Siemensen, H. C.
TI Pharmacokinetics of azathioprine under hemodialysis
SO Int. J. Clin. Pharmacol. Biopharm. (1976), 14(4), 298-302
CODEN: IJCBDX

=> log y

COST IN U.S. DOLLARS SINCE FILE
TOTAL ENTRY
SESSION
FULL ESTIMATED COST 44.31
65.11

STN INTERNATIONAL LOGOFF AT 17:31:11 ON 15 AUG 1998